opposite side bulging, this asymmetry was more marked posteriorly. Chiladiti,[17] in 1910, diagnosed absence of the left lung from a roentgen-ray plate in a boy, aged ten years, who had a flattened left chest. It is possible that his case represents a true absence of one lung. But up to the present the clinical diagnosis of absence of a lung has never been verified at autopsy.

I desire to express my thanks to Dr. Carlos P. Chagas, of the pathological department, for having performed the autopsy and for having placed the literature on the subject at my disposal.

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SECONDARY MENINGITIS TREATED BY INTRASPINOUS ADMINISTRATION OF AUTOGENOUS SERUM.

REPORT OF A CASE.

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During the last epidemic of influenza, some bronchopneumonia cases terminated in a fatal meningitis. These meningitides were reported to have been caused by pneumococci, various kinds of streptococci, and occasionally Bacillus influenzæ.

Two such postinfluenza meningitis cases occurred in my ward at U. S. A. General Hospital No. 12. The second one was treated successfully by the intraspinous administration of autogenous serum. This is the case I am reporting, not only because of the favorable outcome, but also in order to have others try out this therapy.

REPORT OF CASE. Patient, L. S., No. 963, a plumber, was admitted from Camp Polk, N. C., December 10, 1918, to U. S. A. General Hospital No. 12, Biltmore, N. C., in a seemingly moribund condition. A history was not taken at this time because of his irrational and stuporous mental condition. However, on January 4, 1919, the following facts were obtained:

Personal History. About one whisky a day. Smokes fifteen cigarettes per diem. Otherwise negative.

Family History. Father died of sepsis from a carbuncle. Mother, one sister and a brother all alive and well. No history of any hereditary diseases obtained.

Past History. Had trouble with his left ear two years ago and says he does not hear well in that ear. He never had measles, mumps, scarlet fever, diphtheria, smallpox, pneumonia, meningitis or malaria. He denies venereal diseases by name and symptoms.

Present Illness (taken January 4, 1919). Onset of the present illness on December 1, with "a heavy cold," when he "coughed night and day." Also had a high fever. He was told at Camp Polk that he had pneumonia. Was admitted to U. S. A. General Hospital No. 12 on December 10.

Physical Examination. General condition: A dyspneic, cyanotic, toxic, stuporous and moribund white male, aged twenty years.

Skin and mucous membranes: No eruption; face flushed and drawn; eyes and pharynx congested.

Glandular system: No general adenopathy. No thyroid enlargement.

Vascular system: Pulse slow, regular, of good force and collapsible.

Heart: Not enlarged. No adventitious sounds.

Lungs: Bronchopneumonia, bilateral, disseminated (physical signs will appear below).

Genito-urinary System: Faint trace of albumin. Occasional granular cast.

Abdomen: Flaccid.

Liver: Reaches from fourth intercostal space to costal margin.

Spleen: Not palpable. No tenderness. No masses.

Nervous System: Negative except for stupor, which at times slips into semicoma and at times changes to delirium.

Provisional Diagnosis. Bronchopneumonia, disseminated, bilateral, postinfluenzal. Sputum gave a questionable pneumococcus, type 2, precipitation test.

Progress. December 10. Conjunctivæ congested. Laryngitis present. Voice hoarse and husky. Signs of bronchitis in lungs. Too ill to examine thoroughly. Blood-pressure 100/55. Telegram sent: recovery doubtful.

Dec. 11. Condition not improved. Patient delirious. Vomited dark-green fluid. Given intravenously 1000 c.c. of a 5 per cent. glucose solution. We are testing out convalescent influenza, bronchopneumonia donors, who had a type 2 pneumococcus sputum, for a transfusion tomorrow. Blood count: White blood cells, 38,000; polymorphonuclears, 94 per cent.

Dec. 12. Transfusion of 500 c.c. of a convalescent postinfluenza, bronchopneumonia donor given by the sodium-citrate method by Lieut. A. Orth. Blood culture sterile.

Dec. 13. Patient is decidedly improved. Temperature 102°, pulse, 76, respiration, 26; whereas on admission temperature was over 105°, pulse 105, respiration 38. Signs in lungs of a disseminated bronchitis. Although consolidation is not definitely found, it probably exists. (Sputum culture showed Gram-positive diplococci; pneumococcus agglutination test gave a questionable positive type 2 reaction.)

Dec. 14. Condition improved markedly. Temperature 100°, pulse 64, respiration 24. Signs in chest diminishing. Telegram: improving.

Dec. 15. Temperature rose again to 103°. Signs in lungs as they were. Complains of a headache.

Dec. 17. Signs in right lower lobe posteriorly extending as far as the axilla still marked, but no absolute or classical evidence of consolidation. Temperature vacillating. Still complains of a headache.

Dec. 18. Patient is again irrational at times. Cardiovascular system in good condition. Lungs: crepitant and coarse moist rales heard almost everywhere. No definite signs of consolidation, though it probably exists. Nervous system: headache, photophobia, stiff neck, slight bilateral Kernig. Right knee-jerk almost absent. Left knee-jerk diminished. No Babinski, Oppenheim or Gordon. No clonus. Considering the temperature and signs of central nervous system, meningitis is probable. Telegram: recovery still doubtful. Blood culture again sterile.

Dec. 19. Temperature still septic. Blood-pressure 132/78, whereas on admission it was 100/55. Patient appears a little better. Neck still stiff. Kernig doubtful. Knee-jerk diminished. Not as irrational as yesterday. No headache, no hyperesthesia, no photophobia.

Dec. 20. At right base, posteriorly, there is now dulness, higher pitched breathing and crepitant and fine moist rales.

Neck stiff. Headache. Kernig positive. Lumbar puncture should not be deferred any longer. In afternoon lumbar puncture done; 40 c.c. of turbid, cloudy, purulent fluid, under increased tension, obtained.

EXAMINATION OF SPINAL FLUIDS.

Date, 1919.	Cell counts.	Globulin.	Colloidal gold.	Cultures.	Films.	Mouse inoculations.	Sugar.	Appearance.
Dec. 20	61,400 Polys. 93 per cent.	4+	2, 2½, 2, 3, 4, 3, 3, 1, ± 0, 0	No growth	Many Gram-pos. diplococci, intra- and extracellular, resembling pneumococci	Fluid did not kill mouse	No reduction	Purulent.
Dec. 23	....	...	....	No growth	....	......	......	Purulent.
Dec. 25	31,800 per cmm.	...	....	No growth	....	......	......	Purulent.
Dec. 26	....	...	....	No growth	....	With sediment of 20 c.c. of fluid did not kill mouse	......	Purulent.
Dec. 27	415	...	....	No growth	....	......	......	Yellow, less cloudy.
Dec. 28	....	...	....	No growth	Moderate number of pus cells and occasional gram + diplococci. No tuberculosis	No effect on mouse	......	Clearer.
Dec. 30	600	...	....	....	No bacteria found	......	......	Normal tension perceptibility clearer.
Jan. 1	300	...	....	No growth	No bacteria; pus cells; occasional red blood cells	......	......	Clear yellow fluid.
Jan. 2	240	...	....	No growth	....	......	......	Clear yellow.
Jan. 6	20	±	....	No growth	....	......	......	Clear yellow.
Jan. 15	13	±	1½, 2, 2, 2½, 2, 1, 0, 0, 0, 0, 0	No growth	....	......	Reduction	Clear aqueous.

OTHER LABORATORY FINDINGS.

Blood cultures sterile Dec. 12, 18, 21 and 27.

Jan. 12. Pus from parotid duct = Staphylococcus aureus and Streptococcus viridans. Mouse did not die from broth inoculation of the latter.

Sputum: For type of pneumococcus; no definite agglutination. Not bile soluble.

Urine precipitation test for type pneumococcus, negative.

Wassermann, 1 plus.

Blood Counts—Dec. 10: White blood cells, 38,300; polynuclears, 94 per cent.
Dec. 20: White blood cells, 26,900; polynuclears, 90 per cent.
Jan. 4: White blood cells, 21,200; polynuclears, 66 per cent.

Spinal Fluid:

Cell count, 61,400.

Differential count: polymorphonuclears, 93; lymphocytes, 7.

Globulin, 4 plus.

Colloidal gold reaction.

Test-tube No. 1, 2, 3, 4, 5, 6, 7, 8, 9, 10 control.
Result: 2, 2½, 2, 3, 4, 3, 3, 1, ±, 0, 0.

Sugar reduction test, negative.

Culture, no growth.

Original film showed gram + diplococci, both resembling pneumococci, intracellular and extracellular.

Mouse inoculation, mouse remained alive.

Blood count: White blood cells, 26,900; polymorphonuclears, 90 per cent.

Dec. 21 (family present). Lumbar puncture done again this morning; 45 c.c. of cloudy fluid removed. Patient is apparently slightly improved. In afternoon lumbar puncture, 40 c.c. of cloudy fluid under less tension than that previously removed. Retinoscopy: Bilateral edematous papillitis (choked disk). Blood culture, sterile.

Dec. 22. Lumbar puncture done twice today.

Dec. 23. No headache. Neck not so stiff. In afternoon lumbar puncture done; 33 c.c. of cloudy fluid withdrawn. An autogenous serum for intraspinous treatment is being prepared. The patient evidently has immune bodies in his blood, for he is overcoming the pulmonic process. Culture of spinal fluid on blood agar, blood broth and serum broth negative.

Dec. 24. Left lung posteriorly, crepitant and moist rales. Right lung, lower lobe, is still consolidated. At 11 A.M. lumbar puncture. 40 c.c. of pus-laden fluid withdrawn. Injected 15 c.c. of autogenous serum. At 9 P.M. lumbar puncture repeated. 60 c.c. of less cloudy, yellowish fluid drained off. Injected 15 c.c. of autogenous serum.

Dec. 25. Lumbar puncture done twice today and autogenous serum injected. Examination of spinal fluid showed quantity 42 c.c., cell count 31,800, culture again negative.

Dec. 26. Lumbar puncture done and serum given twice today. Mouse inoculation twice with the sediment from 20 c.c. of spinal fluid did not kill the animal.

Dec. 27. Lumbar puncture repeated. 25 c.c. of autogenous serum administered. Spinal fluid is now yellowish from the serum and a little less cloudy perhaps. However, the general condition of the patient is worse. He is irrational. Blood culture again sterile.

Dec. 28. Although the temperature is persistently decreasing under the use of autogenous serum, the general condition of the patient is worse. He is now stuporous and irrational, but the vegetative organs seem to be holding up well. Mouse inoculation with the sediment of 30 c.c. of centrifugalized spinal fluid showed no effect on the mouse. Culture of the spinal fluid on blood agar and blood broth sterile. Smear showed moderate number of pus cells and occasional Gram-positive diplococci. Smear for tuberculosis negative.

Dec. 29. Transfusion of 450 c.c. from a convalescent, post-influenzal, bronchopneumonia donor done by the sodium-citrate method. This was deemed necessary because the patient has been repeatedly bled in order to make up autogenous serum and because of the benefit of the last transfusion. Serum treatment twice today. Temperature has slowly declined until now it vacillates between 99° and 100°.

Dec. 30. Lumbar puncture. Spinal fluid: 600 cells per c.mm. Bacteria negative.

Dec. 31. Temperature normal. Patient appears less stuporous, but he is still irrational. Examination of left lung shows it almost clear and resonant. Lumbar puncture showed the fluid under normal tension and for the first time perceptibly clearer. It has, of course, a yellow hue from the serum. All in all the prognosis is more encouraging. Spinal fluid: cell count, 415 per c.mm.

Jan. 1. Patient is no longer irrational. Neck is not stiff. Kernig, however, still positive. Pupils dilated. Pulse: lower tension and dicrotic. Appetite very good. Lumbar puncture done and 25 c.c. of clear, yellowish fluid removed under normal tension. 20 c.c. of autogenous serum injected into the spinal canal. A clear fluid corroborated the clinical symptoms of marked improvement. Spinal fluid: 300 cells per c.mm. Smear: Bacteria negative, occasional pus cell. Few red blood cells. Culture negative.

Jan. 2. Lumbar puncture, 240 cells per c.mm. autogenous serum administered.

Jan. 3. Still delirious at times, but neck is no longer stiff. Kernig is still positive. Knee-jerk unequal. Lumbar puncture: Canal entered and blood was withdrawn twice. No serum given. Temperature under 100°.

Jan. 4. Neurological and mental status. Cranial nerves are normal. Somatic nerves: no anesthesia, no paralysis. Reflexes; pupils dilated; both react to accommodation. Right pupil reacts sluggishly and slightly to light; left reacts less sluggishly. Bicipital and tricipital reflexes normal. Pectoral, abdominal and cremasteric reflexes normal. Right knee-jerk absent. Left knee-jerk diminished. Neck still slightly stiff. Kernig sign still positive. Achilles and patella reflexes normal. No Babinski, Oppenheim or Gordon.

Perception: Fairly good but a little hazy. He recognizes and knows the use of common objects. He is oriented as to time fairly accurately, but is perfectly oriented as to place and person. No hallucinations exist. He does not dream.

Memory: He recalls well school days, date of enlistment and military life.

Emotionally stable.

Judgment and reason good. In case of fire he says he would wait for help. Were he able he says he would run with water-buckets to the fire. Lately he had been occasionally delirious. Only when delirious from pneumonia, pyrexia and meningitis did he show delusions and confusion. Then he talked about his wife, although he is single. However, he says he is now engaged.

Volition: Rather apathetic. Can feed himself, he says, but nurse does it for him. Involuntary defecation and micturition exists.

Attention: Is good.

Train of thought is coherent.

All in all, he is fast approaching normal mentality. The prognosis for a complete mental recovery is good. Blood count: white blood cells, 21,200. Polymorphonuclears, 66 per cent.

Jan. 5. Patient's temperature around 99°. He feels good. Pulse 100. Patient has bleeding hemorrhoids; a small indurated swelling is presenting itself below the mastoid process, just behind the right ear. Because the pulse has become more rapid, because the patient has been repeatedly bled for autogenous serum, and because of bleeding hemorrhoids and epistaxis from picking his nose, and because of the cervical infection, a blood transfusion of 500 c.c. was again done by the sodium-citrate method.

Jan. 6. Lumbar puncture done. 30 c.c. of rather clear fluid under increased tension removed. 22 c.c. of autogenous serum administered. Spinal fluid: 20 cells per c.mm. Globulin test: slight precipitate. Culture, negative.

Jan. 7. The swelling and inflammation of tissue below the right ear is more superficial and spreading. Urine: Pneumococcus precipitation test; no precipitation.

Jan. 8. Right ear-drum shows a prominent malleus with blood-vessels running down along it. Moreover, all around the margin of the drum vessels are migrating toward the center. Retinoscopy shows choked disk less severe than previously; also what appears to be an exudative retinitis.

Jan. 9. The swelling has grown larger and has spread and involves the whole parotid gland. It is red, hot, indurated and tender. There is no fluctuation. The temperature has risen, but this is due to the parotid inflammation, not the meningitis, which is practically cured. Eyes still staring. Kernig present. The hemorrhoids have bled considerably and he seems to pick at them.

Jan. 10. Swelling has now involved the whole right parotid. On gentle massage a bead of pearly pus presented at the orifice of Stenson's duct.

Jan. 11. Parotitis still the same. Temperature dropped. Kernig still present. Lungs seem clear.

Jan. 12. Parotid not so swollen nor so tender. Temperature is normal. Stenson's duct is still patent and a drop of yellow pus can be pressed through the pouty orifice. Kernig sign is much less marked. Head not stiff. Mind is clear. Culture of pus from Stenson's duct showed a Pneumococcus-streptococcus viridans and a Staphylococcus aureus.

Jan. 13. Swelling of gland is decreasing. Pus can be expressed at will through the oral orifice.

Jan. 14. Parotid swelling decreasing, orifice less pouty and from it drains creamy pus.

Jan. 15. Parotid swelling nearly all gone. Pressure on the gland causes pus to be evacuated through the mouth.

Jan. 23. Lumbar puncture done. 25 c.c. of clear, aqueous solution withdrawn.

Spinal Fluid:

1. 13 cells per c.mm.
2. Globulin: plus-minus.
3. Colloidal gold reaction.
    Test tubes Nos. 1, 2, 3, 4, 5, 6, 7, 8, 9, 10 control.
    Result: $1\frac{1}{2}$, 2, 2, $2\frac{1}{2}$, 2, 1, 0, 0, 0, 0.
4. Sugar, faint reduction.

Jan. 25. Patient feels fine. Appetite is splendid, Bowels regular. He is permitted to go to the bathroom. Temperature, normal.

Neck limber. Parotid swelling completely gone. Kernig negative. Right knee-jerk greatly diminished, only evident when reinforced. Left knee-jerk diminished to a lesser extent.

Retinoscopy: The papillitis and retinitis have disappeared. Fundi now appear normal.

The right ear-drum, which was hyperemic when the parotitis existed, has also returned to its normal appearance.

(These notes are taken verbatim from Clinical Record No. 963-1925, U. S. A. General Hospital No. 12.)

The subsequent notes are condensed and will show an uneventful recovery.

Jan. 31 to Feb. 4. Patient ran a low-grade afternoon temperature, evidently due to slight exertion.

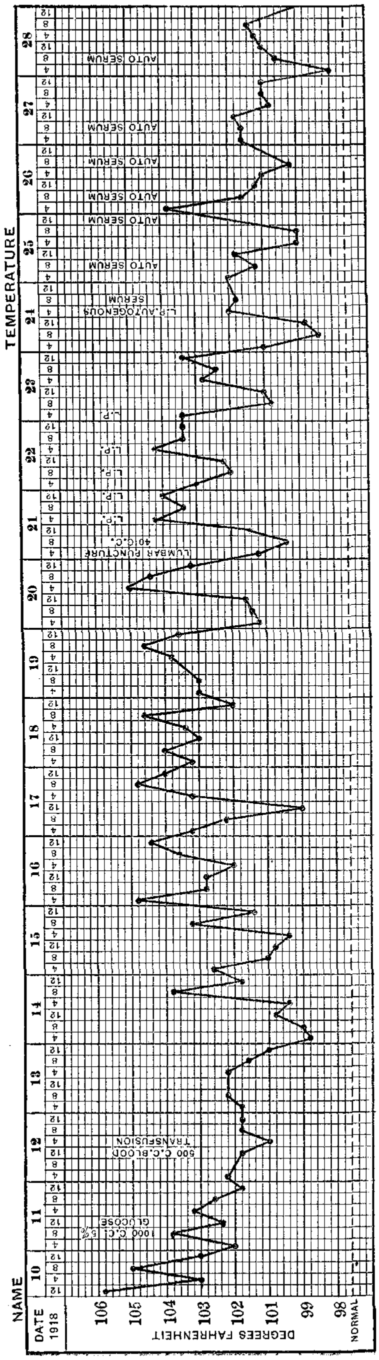
April 10. Neurological and mental examination negative.

April 29. Physical examination showed defective hearing in left ear. He may be discharged on certificate of disability. Meanwhile patient had been on graded exercises, as were all convalescent patients.

A note by a specialist said: Hearing, right ear, 9/20; left ear, 0/200.

Diagnosis: Partial deafness in left ear, probably of central origin and exaggerated as to degree.

DETAILS FOR PREPARATION AND ADMINISTRATION OF AUTOGENOUS SERUM. Aseptically insert a needle into the median basilic vein of the patient at the elbow and draw off blood into four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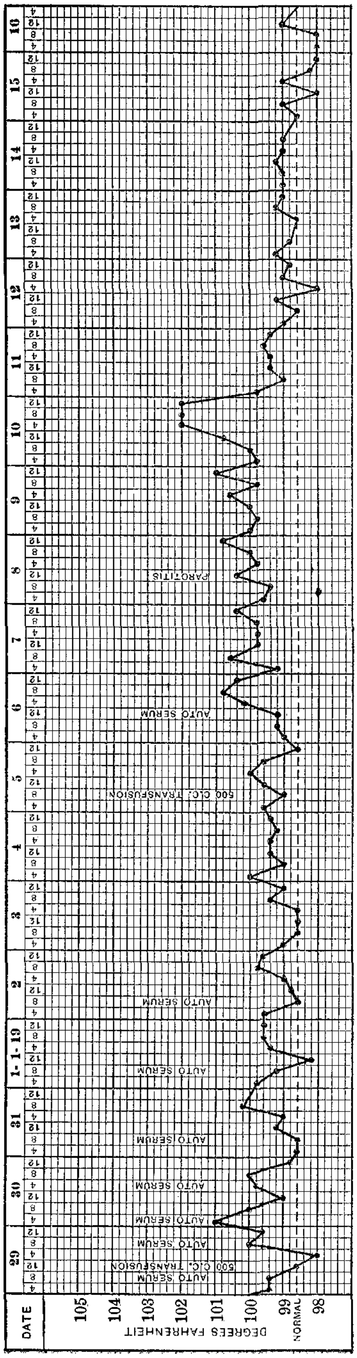
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PAROTITIS

50 c.c. centrifuge tubes. Separate the clot with a flamed platinum needle and centrifuge carefully at low velocity. After the serum is separated the tubes are kept in the ice-box. Centrifuging is not absolutely necessary if the serum separates easily. The spinal canal is tapped in the usual manner with the Quincke needle and spinal fluid is allowed to flow off slowly. About 30 to 60 c.c. of the turbid fluid is drawn off; the amount, of course, depends upon the spinal pressure and the clinical condition of the patient. The clear serum, which had previously been warmed to body temperature, is drawn into a 50 c.c. Luers syringe and injected into the spinal canal. Then the foot of the bed is raised.

Lumbar puncture and administration of serum should be done morning and night until the temperature drops. Then it should be done once a day; eventually on every second day and thus gradually decreased according to the clinical course of the disease and the laboratory examination of the spinal fluid.

DISCUSSION. The rationale of autogenous serum therapy in secondary meningitis is evident for the following reasons:

1. Immune bodies exist in the serum in practically all infectious diseases.
2. This immune serum is brought into direct contact with the inflamed meninges and with the offending microörganisms.
3. Ordinarily the choroid plexus bars the free passage of immune bodies from the blood to the spinal fluid.

The principle suggested itself in this case because the patient was slowly overcoming the pneumonic process when the meningitis occurred. It was logical to suppose that he had immune bodies in his serum.

It is interesting to analyze this case more thoroughly and to evaluate the various procedures.

Upon admission the moribund pneumonic patient was literally revitalized by the timely use of three common therapeutic measures.

1. An infusion of 1000 c.c. of 5 per cent. of glucose solution which caused elimination by diuresis, increased cardiac function and had nutritional value.
2. Immune blood transfusion on the second day, which acted as a stimulant, and as a neutralizer of the toxemia.
3. Cardiac stimulation with caffein sodium benzoate, digitalin and camphor oil, alternatively, every six hours.

The patient, with the help of these therapeutic methods, was successfully combating the pneumonia; the temperature was stepping down; the leukocytosis was high; the whole clinical condition was improving remarkably when the meningitis started.

After the meningeal infection had gained headway, lumbar puncture *per se* was life-saving, because it relieved the otherwise fatal intracranial compression and because it drained the purulent toxic spinal fluid. Whether rachicentesis alone with the natural and transfused immunity would have cured the meningitis, no one can say. The fact is that six days after the treatment with the intraspinous autogenous serum the temperature was normal. Although the patient seemed somewhat better just before the spinal treatment was instituted, we all know how misleading those apparent improvements in meningitis cases actually are; recurrences are the rule. Even from a hypercritical and conservative standpoint we can justly state the result is suggestive. It undoubtedly seems to have been beneficial in this case and may possibly aid in others. It cannot be denied that it is a very rational therapy and therefore should be tried in the absence of a specific immune serum of known value.

The unilateral suppurative parotitis was an unusual complication. The culture of the pus showed a Staphylococcus aureus and a Gram-positive diplococcus belonging to the Pneumococcus-streptococcus viridans group. This was probably not a hematogenous but an ascending duct infection, as four blood cultures were negative. The orifice of the duct was always patent and always drained pus until resolution occurred without surgical interference. No apparent parenchymatous destruction of the gland took place.

Three 500 c.c. transfusions were given. All the donors were convalescent influenza Bronchopneumonia patients. All were infected, as was this patient, at Camp Polk. The first transfusion was given to combat the pneumonia and the concomitant toxemia. The second was given to make up the blood withdrawn to prepare the autogenous serum. The third was given for the same reason and because blood was lost by epistaxis and bleeding hemorrhoids; then, too, it cut down the convalescence and may have helped the parotitis.

Laboratory Discussion. I have delayed publishing this article because I should have preferred to have had Capt. A. L. Garbat, M. D., New York City, chief of the laboratory, discuss this subject. However, he has been ill and hence unable to contribute his part.

It was unfortunate that no absolute conclusion could be reached as to the type of organism we were dealing with in the meningeal infection. The sputum gave a doubtful capital group 2 agglutination by the mouse method, but the organisms were not bile-soluble. The urine showed no precipitation tests. Repeated cultures from the turbid spinal fluid made on all types of plain blood or serum media remained sterile. Repeated animal inoculations with the sediment of the spinal fluid did not kill the mice, although stained specimens of the sediment showed definite Gram-positive diplococci both intra- and extracellular which had the morphology of pneumococci. Apparently the bacteria were dead. Smears from the pus of the parotid showed a similar organism, but these bacteria grew rapidly on artificial media, with the characteristics of a Streptococcus viridans. The cultures produced green pigment on blood agar, were bile-insoluble, were not agglutinated by pneumococcus serum and did not kill mice. Naturally one cannot draw any definite conclusions as to the nature of the meningeal infection from these findings. This does not, however, materially influence the point of importance which the author desired to bring out, namely, that it is wise in secondary meningitis to use intraspinally the autogenous serum, which probably contains a number of antibodies from the primary infection.

CONCLUSION. We recommend the use of autogenous serum injected into the subarachnoid space in cases of secondary meningitis when there is no efficient specific immune serum.

I must take this opportunity to thank Capt. A. L. Garbat, chief of the laboratory of U. S. A. General Hospital No. 12 for his ever-helpful coöperation. I also appreciate the assistance given by Sgt. P. Piel and his brother Pvt. R. Piel, of the laboratory force, who carefully made up the autogenous serum under our direction.

OBSERVATIONS ON THE COLLOIDAL GOLD REACTION WITH CEREBROSPINAL FLUID.

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DURING the past four years the colloidal gold test on cerebrospinal fluid as devised by Carl Lange has aroused great interest among physicians and laboratory workers. This reaction, the exact nature of which is not yet clearly understood, is proving of some practical value as a diagnostic procedure. The utility of the test is not generally recognized at the present time, and frequently the reaction yields information not obtained so quickly by any other diagnostic measure. This is seen particularly in the occasional case of meningitis in which the Wassermann reaction is negative and a characteristic curve is obtained by the colloidal gold method, or where a tuberculous curve is obtained and no acid-fast bacilli found; also in cases in which differential diagnosis between poliomyelitis and tuberculous meningitis is difficult the reaction has proved of value. As in all laboratory diagnostic procedures, however, occasional false reactions are obtained, and it is the chief object of this paper to record a few such instances and to make certain observations which may prove of interest to those performing the test.

Previous articles on the gold reaction have considered in great detail the methods of preparing the fluid, and we shall not discuss the subject except to say that if directions regarding cleanliness of glassware and purity of reagents are carefully adhered to no great